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Travel-Related Changes in Physiological Health Markers: The Effects of Transmeridian Travel on Female Volleyball Players

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Abstract

International Journal of Exercise Science 19(2): 2006, 2026. This study investigated the impact of stress induced by repeated transmeridian travel, with particular emphasis on transmeridian time-zone crossings that compress daylight hours and disrupt circadian rhythms, on the health physiology of female volleyball athletes. Research compared pre-/post-season changes in forty-three travel-team (age 20.3±1.38) and twenty-six non-traveling (age 19.52±0.97) players from the University of Hawai'i at Hilo (UHH). Pre-season data were collected before the first travel game, with postseason data collected 15 weeks later. Data were collected using an OMRON M4-1 IntelliSense monitor, SECA 200 tape measure, GE Prodigy Lunar DXA, and salivary ELISA assays. Analyses of data were conducted using SPSS to run repeated-measures ANOVA. Significant travel-group pre- to post-season mean changes were observed in resting heart rate (57.36±7.91 vs. 63.95±8.12 bpm, respectively; $p < 0.001$), systolic blood pressure (110.27±6.76 vs. 114.50±6.70 mmHg, $p < 0.05$), diastolic blood pressure (65.28±6.03 vs. 70.08±7.03 mmHg, $p < 0.001$), hip circumference (96.49±7.83 vs. 93.97±8.04 cm, $p < 0.01$), waist circumference (76.61±7.59 vs. 78.67±7.52 cm, $p < 0.01$), trunk body fat deposition (27.82±6.67 vs. 30.72±6.94 %, $p < 0.001$), salivary cortisol (13.24±5.52 vs. 25.79±14.02 nmol/L, $p < 0.01$), and leg bone mineral density (1.427±0.11 vs. 1.459±0.11 g/cm2, $p < 0.01$). Other variables investigated in this study were not significantly different. Findings suggest that the extended intermittent stress associated with circadian rhythm disruptions throughout the volleyball season may meaningfully influence physiological health markers.

Keywords: Body composition, fat redistribution, circadian rhythm, intermittent stress, travel-induced stress

Introduction

The circadian rhythm in the human body represents the output of the suprachiasmatic nucleus (SCN), which serves as the central pacemaker. Through its neurohumoral signaling, the SCN synchronizes daily 24-hour cycles in physiological processes to external time cues such as light, partly through hormonal signaling, including melatonin release from the pineal gland.[1-2] Cortisol follows a circadian secretion pattern regulated by the hypothalamic-pituitary-adrenal (HPA) axis and plays a central role in the stress response.[3] Within this axis, the hypothalamus secretes corticotropin-releasing hormone (CRH), stimulating the anterior pituitary gland to release adrenocorticotropic hormone (ACTH), which in turn promotes cortisol production by the adrenal glands. Elevated cortisol levels inhibit both CRH and ACTH release through a negative feedback loop.[4] Prolonged disruption of circadian entrainment may dysregulate HPA

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axis activity, contributing to altered cortisol rhythms and increased vulnerability to chronic and abnormal stressors.[5]

Transmeridian air travel, particularly repeated eastward flights, imposes large circadian phase shifts that challenge bodily homeostasis and the internal circadian rhythm.[6] Traveling from west to east compresses daylight exposure and requires phase shift advances, to which the body adapts more slowly than the phase delays induced by westward travel.[4] Overexposure to artificial light during air travel may additionally impede sleep and exacerbate stress levels.[7-8] Frequent travelers can experience issues such as prolonged circadian disruptions, associated with health concerns such as cardiovascular diseases, metabolic disorders, compromised immune function, and a heightened risk of sleep disorders.[9-10] Investigating the effects of travel-induced circadian rhythm disruptions can enhance the current understanding of how intermittent stress impacts physiological markers, including body fat distribution, blood pressure, resting heart rate, bone mineral density, and cortisol levels.

Athletes who frequently cross time zones are vulnerable to circadian misalignment, sleep disruption, and heightened stress loads from rigorous physical routines. Our study sample, the University of Hawai'i at Hilo (UHH) women's volleyball team, naturally travels eastward more frequently than most teams nationwide. The travel team logs an approximate 16,000 miles per 15-week volleyball season. In addition to the academic, social, and travel pressures that challenge most collegiate athletes, UHH women's volleyball players face amplified stressors from frequent transmeridian air travel. Flying from the Hawaii time zone (HST) to the Pacific Time Zone (PST) and Mountain Time Zone (MST) causes significant time zone shifts and circadian rhythm adjustments, due to a minimum time difference of two hours and maximum of four hours.

Prior research has primarily focused on short-term effects of jet lag on performance metrics (e.g. reaction time, agility, in-game performance), while the chronic health consequences of repeated travel across a competitive season, correlated to circadian rhythms, remain poorly understood.[11-12] In particular, little is known about how frequent travel-induced circadian disruptions impact cardiovascular health, body fat distribution, bone mineral density, and cortisol regulation in athletes. Most studies evaluating athletes' travel have emphasized competition outcomes rather than physiological well-being.[13-14] Chronic circadian misalignment has been implicated in metabolic disorders, cardiovascular disease risk, and altered adipose distribution in shift-work and general population studies, suggesting potential parallel risks for athletes exposed to recurrent transmeridian travel.[15-17] Evidence linking these physiological measures and risk outcomes is scarce, and few studies to our knowledge have sufficiently evaluated bone mineral density in this context, despite its relevance to both health and performance.

This study addresses a critical gap in the literature by evaluating the physiological consequences of repeated transmeridian travel in female collegiate athletes, a population that has been largely overlooked in travel-related health research. Cardiovascular measures, body composition, regional bone mineral density, and salivary cortisol were examined to assess the chronic impact of travel-associated circadian disruption and stress. Salivary cortisol was evaluated as an indicator of circadian disruption, which is commonly observed following eastward travel, and of physiological stress levels in the study participants. The findings of this study may hold broader relevance for non-athletic populations who experience frequent travel-related circadian misalignment. We hypothesized that athletes exposed to regular transmeridian travel and resulting circadian disruptions would exhibit more significant increases in visceral trunk fat, cortisol levels, resting

heart rate, mean arterial pressure, and both diastolic and systolic blood pressure over the course of the competitive season compared with non-traveling teammates.

Methods

Participants

The study compared baseline and post-season physiological health markers and salivary cortisol levels between transmeridian traveling and non-traveling collegiate volleyball players to evaluate the chronic physiological effects of travel-associated circadian disruption. The travel group consisted of athletes routinely exposed to transmeridian air travel across time zones, whereas the control group consisted of non-traveling teammates who maintained the same training regimen but did not experience travel-related stressors. This design allowed between-group physiological differences to be interpreted as primarily associated with travel exposure independent of training load effects.

The UHH women's volleyball team provided a cohort of young, physically active, and medically cleared athletes, ensuring a relatively homogeneous and healthy sample at baseline across both groups. This allowed observed changes in cardiovascular, endocrine, and body composition outcomes to be interpreted within a population not typically expected to exhibit statistically significant adverse physiological change in the absence of external stressors such as travel. Sample size was not predetermined due to anticipated annual fluctuations in team roster size. Forty-three Division II travel-team (WVB) university women volleyball players (mean age 20.3 ± 1.38 years) and twenty-six control group non-travel (CT) university women volleyball players (mean age 19.52 ± 0.97 years) were included.

The study commenced at the beginning of the volleyball season and concluded approximately 15 weeks later, corresponding to the competitive season timeline. Athletes who participated in multiple seasons were only included once in the dataset; specifically, the first season in which an athlete was enrolled during the study period was used for analysis. This ensured that each participant contributed only a single data point, regardless of total seasons played, to maintain statistical independence.

Written informed consent was obtained from all the participants prior to each data collection period. An overall participation rate of at least 80% in seasonal competitions was required for travel-group study participants. No distinction was made between starters and non-starters. The control group (CT) and travel-team (WVB) volunteers were recruited from the same athletic team; however, CT athletes did not undergo athletics-related travel throughout the season. During periods when the travel group competed in away games, control athletes continued supervised training of comparable intensity overseen by the same coaching staff. As red-shirt athletes, CT participants also engaged in at-home practice games, ensuring comparable training volumes across groups aside from travel exposure.

The above methodology was repeated across four separate seasons. This study was approved by The Institutional Review Board (UH IRB) of the University of Hawaii system (No. 2023-00349) for the course of each volleyball season. This research was carried out fully in accordance with the ethical standards of the *International Journal of Exercise Science*.[18]

Protocol

Both the WVB and CT volunteers were measured immediately before the women's volleyball team competed in their first seasonal game (T1). Baseline salivary cortisol was collected prior to the start of the competitive season to establish pre-season endocrine status. The post-season assessment (T2) was conducted approximately 10 days after the final game of the season to minimize confounders from acute exercise effects. This interval was chosen to allow cardiovascular, hormonal, and body composition measures to return closer to baseline resting levels, limiting the influence of competition-related fatigue. Both travel and non-travel athletes were assessed on this same timeline, ensuring comparable conditions across groups. The time of testing was kept constant for each participant at T1 and T2, with the majority of measurements collected between 10:00 AM HST and 12:00 PM HST. Participants were instructed to replicate pre-testing sleep and dietary patterns from T1 prior to T2 testing. All physiological measures were obtained as resting assessments, conducted in the morning prior to any training or exercise. Participants rested supine for 3 to 5 minutes before assessment, and cardiovascular measurements were recorded in triplicate for reliability.

Heights and weights were measured using a digital scale and stadiometer (SECA 769, Hamburg, Germany) that was calibrated pre-test. Participants wore light, fitted clothing to ensure consistent data collection, with clothing type remaining constant across each collection period. Circumference measurements were performed with a SECA 200 tape measure. Thigh circumference was measured on the dominant side non-weight-bearing-appendage at the midpoint between the superior patella and the greater trochanter. Waist circumference was measured horizontally upon expiration at the midpoint between the superior iliac crest and the inferior point of the costal cartilage. Hip circumference was obtained at the level of the greater trochanters with modulations made to include the largest horizontal circumference. To ensure data accuracy and subject comfort, all circumference tests were conducted in front of a mirror and with two female technicians present.

Body composition analyses and bone mineral density measurements were collected via GE Prodigy Lunar DXA (dual energy X-ray absorptiometry) unit (GE Medical Systems Luna, Madison) with the enCORE software (v. 16.2). The scanner was calibrated before each data collection period. Participants completed a whole-body DXA scan that provided a three-component analysis of body composition. Total regional bone mineral densities and lean and fat mass depositions were assessed. Measurements were provided in grams of tissue types and percentages of body fat and lean mass for both whole body as well as regional segments including the arms, legs, and trunk. Participants were positioned supine, with hands by their sides.

Blood pressure (BP) and heart rate (HR) measurements were obtained using a fully automatic monitor (OMRON M4-1 IntelliSense). Participants rested in a supine position for 10 minutes prior to the initial measurement, with a subsequent measurement taken 3 minutes later. Measurements of diastolic blood pressure (DBP), systolic blood pressure (SBP), and HR were averaged if they varied by less than 5% between measurements. Variances that exceeded 5% demanded a re-measurement following an additional 3-minute rest period. Several instances required multiple rest periods to obtain finalized measurements.

Saliva samples were collected using the drool-split method. Participants received strict instructions to abstain from eating, drinking, or oral hygiene procedures for at least 1 hour prior to sample collection. Whole saliva (≥ 10 mL) was collected into test tubes and stored at -80 °C until analysis.

Cortisol levels were determined using commercially available cortisol EIA kits (Salimetrics, State College, PA). The CV% for the assays was <7%.

Statistical Analysis

Statistical analysis was performed using IBM SPSS Version 29.0.2.0 (20). Repeated-measures ANOVA were used to assess within-subject (pre- vs. post-season) and between-group (travel vs. non-travel) mean changes. Potential confounding variables, including age, height, and body weight, were evaluated as covariates in preliminary models; however, none demonstrated significant influence on the main outcomes and were therefore excluded from the final tables. Pairwise comparisons were conducted following significant main or interaction effects to examine within-group changes (pre vs. post within travel or non-travel) and between-group differences at each time point (travel vs. non-travel at pre- and post-season). Effect sizes were included alongside these comparisons to provide additional context. Within-group changes were quantified using Cohen's d whereas between-group differences at each time point were assessed using Hedges' g, which provides a bias-corrected estimate appropriate for unequal group sizes. Simple effects partial eta squared (η^2□) values were reported to quantify the proportion of variance explained by group or time effects. Data are presented as mean ± SD. Statistical significance was set at $p < 0.05$, with $p \leq 0.01$ denoting more robust effects where applicable.

Effect size magnitudes were interpreted according to conventional thresholds. For within-group comparisons using Cohen's d and between-group comparisons using Hedges' g, values of 0.20, 0.50, and 0.80 were interpreted as small, medium, and large effects, respectively. Partial eta squared (η^2□) values were interpreted as small (0.01), medium (0.06), and large (≥ 0.14). These benchmarks were applied to aid interpretation of the practical magnitude of effects rather than for inferential decisions.

Results

The sixty-nine women who participated in the study were relatively homogeneous in age, with the travel group averaging 20.3±1.38 years and the non-travel group 19.52±0.97 years. At the study outset, the travel group had an average height (Ht) of 173.67±7.99 cm, while the non-travel group averaged 172.52±7.07 cm. Additionally, the overall weight (Wt) of the travel group was 70.95±11.05 kg with the non-travel group averaging 69.43±11.77 kg. No significant changes in height or weight were observed across the study period in either group. See Table 1 for descriptive statistics.

Table 1. Statistical characteristics of the Height (Ht) and Weight (Wt) in female college volleyball athletes before and after a season, stratified by travel or non-travel status.

	Travel (n = 43)		Non-Travel (n = 26)		ANOVA		
	pre	post	pre	post	time	group	time x group
Age (yrs)	20.3±1.38		19.52±0.97				
Ht (cm)	173.67±7.99	173.54±8.08	172.52±7.07	172.52±7.26	0.544	0.573	0.566
Wt (kg)	70.95±11.05	70.75±11.11	69.43±11.77	68.98±11.67	0.218	0.559	0.628

Values: mean ± SD; repeated-measures ANOVA [time, group, time × group]. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

No significant changes in weight were found between groups. Statistically significant effects, however, were found in other cardiovascular measurements including systolic blood pressure (SBP), diastolic blood pressure (DBP), and resting heart rate (RHR). The travel group participants

displayed significant increases in the SBP (110.27±6.76 mmHg to 114.50±6.70 mmHg), DBP (65.28±6.03 mmHg to 70.08±7.03 mmHg), and RHR (57.36±7.91 bpm to 63.95±8.12 bpm) measures (Table 2). No such increases were found in the non-travel group. Changes in mean arterial pressure were observed in both groups but did not appear significantly impacted by travel status. See Table 2 for the list of interactions.

Table 2. Selected cardiovascular physiological parameters before and after a collegiate volleyball season, stratified by travel or non-travel status.

	Travel (n = 43)		Non-Travel (n = 26)		ANOVA		
	pre	post	pre	post	time	group	time x group
Age (yrs)	20.3±1.38		19.52±0.97				
Ht (cm)	173.67±7.99	173.54±8.08	172.52±7.07	172.52±7.26	0.544	0.573	0.566
Wt (kg)	70.95±11.05	70.75±11.11	69.43±11.77	68.98±11.67	0.218	0.559	0.628

Values: mean ± SD; repeated-measures ANOVA [time, group, time × group]. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

Pairwise comparisons revealed significant within-group increases in cardiovascular parameters among the travel group. Specifically, systolic blood pressure (SBP) increased by 4.23 mmHg ($p < 0.01$, $d = 0.63$), diastolic blood pressure (DBP) by 4.80 mmHg ($p < 0.001$, $d = 0.73$), mean arterial pressure (MAP) by 4.79 mmHg ($p < 0.001$, $d = 0.88$), and resting heart rate (RHR) by 6.59 bpm ($p < 0.001$, $d = 0.82$) from pre- to post-season (Table 3). In contrast, the non-travel group demonstrated smaller, non-significant pre- to post-season changes across these measures. Between-group comparisons indicated no significant differences at pre-season for SBP, DBP, MAP, or RHR. At post-season, the travel group demonstrated significantly higher RHR values relative to the non-travel group ($\Delta M = +4.15$ bpm, $p = 0.028$*, $g = 0.55$). See Table 3 for the list of interactions.

Table 3. Pairwise comparisons of cardiovascular physiological parameters across time points (Pre → Post) and travel status (Travel vs. Non-travel) in collegiate volleyball athletes.

Variables	Travel Pre → Post	Non-Travel Pre → Post	Travel vs Non-Travel @ Pre (T1)	Travel vs Non-Travel @ Post (T2)
SBP (mmHg)	4.2** [1.44, 7.03], 0.63, 0.063	2.0 [-1.60, 5.59], 0.32, 0.009	-0.9 [-4.06, 2.37], -0.13, 0.002	1.4 [-1.83, 4.61], 0.22, 0.005
DBP (mmHg)	4.8*** [2.39, 7.21], 0.73, 0.104	1.5 [-1.62, 4.58], 0.40, 0.007	-0.8 [-3.53, 2.03], -0.14, 0.002	2.6 [-0.21, 5.35], 0.42, 0.024
MAP (mmHg)	4.8*** [2.62, 6.97], 0.88, 0.124	4.0** [1.18, 6.78], 0.89, 0.056	-0.4 [-2.87, 2.14], -0.08, 0.001	0.5 [-2.05, 2.95], 0.09, 0.001
RHR (bpm)	6.6*** [3.39, 9.79], 0.82, 0.110	0.8 [-3.34, 4.88], 0.12, 0.001	-1.7 [-5.36, 2.01], -0.22, 0.006	4.2* [0.46, 7.83], 0.55, 0.036

Values: ΔM; CI; Cohen's d [Pre → Post]; Hedges' g [@ Pre (T1) or @ Post (T2)]; η^2□. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

No significant changes were found in leg body fat percentage or waist-to-hip circumference ratio between the travel and non-travel groups. Over the course of the study, the observed interactions between time and group produced significant mean differences in measurements including trunk body fat percentage ($p < 0.001$), hip circumference ($p = 0.004$), and waist circumference ($p = 0.009$). The mean changes observed in the non-travel group were statistically less than those of the travel group, yielding non-significant results (Table 4). No significant change was observed in total body fat percentage after the 15-week study period. The interactions are shown in Table 4.

Table 4. Selected body composition indicators before and after a collegiate volleyball season, stratified by travel or non-travel status.

	Travel (n = 43)		Non-Travel (n = 26)		ANOVA		
	pre	post	pre	post	time	group	time x group
Total BF (%)	29.71±6.27	29.50±5.95	30.23±6.40	29.85±5.83	0.187	0.772	0.715
Trunk BF (%)	27.82±6.67	30.72±6.94	28.54±6.65	29.39±6.14	< 0.001***	0.854	< 0.001***
Leg BF (%)	30.97±5.88	28.89±5.44	31.60±5.95	30.09±5.57	< 0.001***	0.515	0.244
Arm BF (%)	33.03±7.22	31.95±7.26	32.37±6.85	31.63±6.60	0.003**	0.775	0.573
MT (cm)	53.20±4.11	51.99±3.93	52.40±3.19	51.38±3.63	0.002**	0.423	0.791
Hip Circ (cm)	96.49±7.83	93.97±8.04	95.55±8.41	94.94±8.85	< 0.001***	0.993	0.004**
Waist Circ (cm)	76.61±7.59	78.67±7.52	77.10±8.09	77.54±8.26	< 0.001***	0.867	0.009**
W/H Ratio (cm)	79.45±5.15	83.82±5.13	80.35±5.17	84.22±5.03	< 0.001***	0.582	0.617

Values: mean ± SD; repeated-measures ANOVA [time, group, time × group]. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

Pairwise comparisons displayed significant within-group changes in body composition among the travel team. From pre- to post-season, significant increases were observed in trunk body fat percentage (ΔM = +2.90%, p = 0.046, d = 0.43) and waist-to-hip ratio (ΔM = +4.37%, p < 0.001, d = 0.85). The non-travel group demonstrated a smaller, significant within-group pre- to post-season increase in waist-to-hip ratio (ΔM = +3.87%, p < 0.001, d = 0.76). Non-significant reductions were observed in leg body fat percentage (ΔM = –2.08%, d = –0.37), arm body fat percentage (ΔM = –1.08%, d = –0.15), mid-thigh circumference (ΔM = –1.21 cm, d = –0.30), and hip circumference (ΔM = –2.52 cm, d = –0.32). Between-group comparisons demonstrated no significant differences in pre- or post-season body composition measures. See Table 5 for the list of interactions.

Table 5. Pairwise comparisons of body composition indicators across time points (Pre → Post) and travel status (Travel vs. Non-travel) in collegiate volleyball athletes.

Variables	Travel Pre → Post	Non-Travel Pre → Post	Travel vs Non-Travel @ Pre (T1)	Travel vs Non-Travel @ Post (T2)
Total BF (%)	-0.22 [-2.83, 2.39], -0.04, 0.000	-0.39 [-3.74, 2.97], -0.06, 0.000	-0.52 [-3.53, 2.48], -0.08, 0.001	-0.36 [-3.36, 2.65], -0.06, 0.000
Trunk BF (%)	2.90* [0.058, 5.74], 0.43, 0.029	0.85 [-2.80, 4.50], 0.13, 0.002	-0.72 [-3.99, 2.55], -0.11, 0.001	1.33 [-1.94, 4.60], 0.20, 0.005
Leg BF (%)	-2.08 [-4.51, 0.35], -0.37, 0.021	-1.51 [-4.64, 1.62], -0.26, 0.007	-0.63 [-3.43, 2.17], -0.11, 0.001	-1.20 [-4.00, 1.60], -0.22, 0.005
Arm BF (%)	-1.08 [-4.09, 1.93], -0.15, 0.004	-0.74 [-4.61, 3.13], -0.11, 0.001	0.67 [-2.80, 4.13], 0.09, 0.001	0.33 [-3.14, 3.80], 0.05, 0.000
MT (cm)	-1.21 [-2.83, 0.42], -0.30, 0.016	-1.02 [-3.11, 1.07], -0.30, 0.007	0.80 [-1.07, 2.67], 0.21, 0.005	0.62 [-1.25, 2.49], 0.16, 0.003
Hip Circ (cm)	-2.52 [-6.02, 0.98], -0.32, 0.015	-0.61 [-5.11, 3.89], -0.07, 0.001	0.94 [-3.09, 4.97], 0.12, 0.002	-0.98 [-5.01, 3.06], -0.12, 0.002
Waist Circ (cm)	2.07 [-1.26, 5.39], 0.27, 0.011	0.45 [-3.83, 4.72], 0.05, 0.000	-0.49 [-4.32, 3.34], -0.06, 0.000	1.13 [-2.70, 4.96], 0.15, 0.003
W/H Ratio (cm)	4.37*** [2.19, 6.56], 0.85, 0.105	3.87** [1.06, 6.68], 0.76, 0.053	-0.90 [-3.42, 1.62], -0.17, 0.004	-0.40 [-2.92, 2.12], -0.08, 0.001

Values: ΔM; CI; Cohen's d [Pre → Post]; Hedges' g [@ Pre (T1) or @ Post (T2)]; η^2□. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

Table 6. Total and regional bone mineral densities from whole body DXA scans before and after a collegiate volleyball season, stratified by travel or non-travel status.

	Travel (n = 43)		Non-Travel (n = 26)		ANOVA		
	pre	post	pre	post	time	group	time x group
T BMD (g/cm2)	1.239±0.07	1.237±0.07	1.226±0.08	1.232±0.08	0.307	0.613	0.104
Pelvis BMD (g/cm2)	1.322±0.10	1.321±0.10	1.311±0.10	1.301±0.10	0.266	0.524	0.344
Arm BMD (g/cm2)	1.129±1.11	0.915±0.14	0.908±0.07	0.933±0.06	0.400	0.355	0.286
Leg BMD (g/cm2)	1.427±0.11	1.459±0.11	1.408±0.09	1.418±0.11	< 0.001***	0.251	0.005**
Spine BMD (g/cm2)	1.146±0.10	1.145±0.10	1.100±0.11	1.099±0.12	0.674	0.085	1.000

Values: mean ± SD; repeated-measures ANOVA [time, group, time × group]. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

A significant time effect was observed for leg bone mineral density (LBMD), as indicated by the ANOVA results (p = 0.005). The non-travel group displayed an initial LBMD mean of 1.408±0.09 g/cm² and a LBMD mean of 1.418±0.11 g/cm² at the end of the study. The travel group mean leg bone mineral density was 1.427±0.11 g/cm² at the study outset and 1.459±0.11 g/cm² at the postseason collection interval. No significant mean changes were observed in the total, pelvis, arm, or spine bone mineral density measurements. The regional bone mineral density interactions are shown in Table 6.

Pairwise comparisons indicated no significant changes in total or regional bone mineral density (BMD) across the season. Neither the travel nor non-travel group demonstrated significant within-group changes from pre- to post-season, and no between-group differences were observed at either timepoint (all p > 0.05; Table 7). A small, non-significant pre- to post-season within-group increase in LBMD was observed in the travel group (ΔM = +0.032, p = 0.173, d = 0.28). See Table 7 for the list of interactions.

Table 7. Pairwise comparisons of total and regional bone mineral densities from whole body DXA scans across time points (Pre → Post) and travel status (Travel vs. Non-travel) in collegiate volleyball athletes.

Variables (g/cm2)	Travel Pre → Post	Non-Travel Pre → Post	Travel vs Non-Travel @ Pre (T1)	Travel vs Non-Travel @ Post (T2)
T BMD	-0.002 [-0.033, 0.030], -0.02, 0.000	0.007 [-0.034, 0.047], 0.08, 0.001	0.013 [-0.023, 0.050], 0.18, 0.004	0.005 [-0.031, 0.041], 0.07, 0.001
Pelvis BMD	-0.001 [-0.042, 0.041], -0.01, 0.000	-0.010 [-0.063, 0.043], -0.10, 0.001	0.011 [-0.037, 0.058], 0.11, 0.001	0.020 [-0.028, 0.067], 0.20, 0.005
Arm BMD	-0.214 [-0.481, 0.054], -0.27, 0.018	0.025 [-0.319, 0.370], 0.39, 0.000	0.221 [-0.087, 0.530], 0.25, 0.015	-0.018 [-0.326, 0.291], -0.15, 0.000
Leg BMD	0.032 [-0.014, 0.077], 0.28, 0.014	0.010 [-0.049, 0.068], 0.10, 0.001	0.020 [-0.033, 0.072], 0.19, 0.004	0.041 [-0.011, 0.094], 0.38, 0.018
Spine BMD	-0.002 [-0.047, 0.044], -0.02, 0.000	-0.002 [-0.060, 0.057], -0.01, 0.000	0.046 [-0.007, 0.098], 0.38, 0.022	0.046 [-0.007, 0.098], 0.42, 0.022

Values: ΔM; CI; Cohen's d [Pre → Post]; Hedges' g [@ Pre (T1) or @ Post (T2)]; η^2□. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

Significant intergroup mean changes were observed in salivary cortisol levels (nmol/L). The travel group showed a significant increase in mean cortisol levels (13.24±5.52 nmol/L to 25.79±14.02 nmol/L). The non-travel group showed no statistically significant change in mean cortisol levels, displaying a marginal increase from 15.49±4.48 nmol/L to 16.48±5.18 nmol/L. A significant time-by-group interaction effect (p = 0.007) was observed in the salivary cortisol measurement. The interactions are shown in Table 8.

Table 8. Salivary cortisol levels from assayed cortisol EIA kits (CV% <7%) before and after a collegiate volleyball season, stratified by travel or non-travel status.

	Travel (n = 43)		**Non-Travel (n = 26)**		**ANOVA**		
	pre	post	pre	post	time	group	time x group
Cortisol (nmol/L)	13.24±5.52	25.79±14.02	15.49±4.48	16.48±5.18	0.002**	0.273	0.007**

Values: mean ± SD; repeated-measures ANOVA [time, group, time × group]. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

Table 9. Pairwise comparisons of salivary cortisol levels from assayed cortisol EIA kits (CV% <7%) across time points (Pre → Post) and travel status (Travel vs. Non-travel) in collegiate volleyball athletes.

Variables	Travel Pre → Post	Non-Travel Pre → Post	Travel vs Non-Travel @ Pre (T1)	Travel vs Non-Travel @ Post (T2)
Cortisol (nmol/L)	12.55** [5.28, 19.82], 1.18, 0.243	0.99 [-6.63, 8.61], 0.21, 0.002	-2.24 [-9.69, 5.21], -0.44, 0.010	9.32* [1.87, 16.76], 0.86, 0.144

Values: ΔM; CI; Cohen's d [Pre → Post]; Hedges' g [@ Pre (T1) or @ Post (T2)]; η^2□. Significance: *p < 0.05; **p < 0.01; ***p < 0.001

Pairwise comparisons revealed a significant within-group increase in salivary cortisol for the travel group (ΔM = 12.55 nmol/L, p < 0.01, d = 1.18). No significant pre- to post-season changes were observed in the non-travel group. Between-group comparisons showed no differences at pre-season; however, at post-season, the travel group displayed significantly higher cortisol levels compared to the non-travel group (ΔM = 9.32 nmol/L, p = 0.016, g = 0.86; Table 9).

Discussion

This study suggests that travel-related circadian rhythm disruptions may contribute to physiological strain in female collegiate volleyball players. The study compared the mean changes in physiological markers between travel-team and non-traveling team volunteers over a 15-week period. The most notable effects were moderate-to-large increases in cardiovascular strain (resting heart rate, systolic and diastolic blood pressure) and heightened salivary cortisol levels in the travel group, while the non-travel group exhibited non-significant changes. Additionally, shifts in regional adiposity, particularly in trunk fat and waist-to-hip ratio, were of note. Consistent with prior literature, these findings suggest that the added burden of repeated transmeridian travel, specifically eastward time-zone crossings, with associated circadian misalignment, places unique stress on athletes that extends beyond the demands of training and competition alone.[19] In contrast, changes in body composition and bone mineral density were generally small or negligible, aligning with their weak or non-significant effect sizes. Collectively, these results emphasize that the cardiovascular and endocrine systems appear vulnerable to travel-related stress, highlighting the need for strategies to mitigate these effects during competitive seasons.

Circadian misalignment and sleep disruption are established contributors to diminished cardiovascular health and the progression of cardiovascular disease (CVD).[20] In this study, the travel group showed significant increases in SBP, DBP, and RHR (Table 2, Table 3). Such acute elevations in resting cardiovascular parameters have been linked to greater risk of metabolic abnormalities and CVD.[21-22] An elevated risk of cardiovascular disease and metabolic abnormalities is typically associated with reduced physical fitness,[23] making the presence of such cardiovascular strain indicators in active collegiate athletes particularly concerning. The observed moderate-to-large effect sizes strengthen the interpretation that these changes were clinically meaningful rather than artifacts of sample size. Notably, at the post-season timepoint (T2), RHR

remained significantly higher in the travel group compared to the non-travel group. The control group consistently did not reach significance across cardiovascular parameters, suggesting that while routine participation in collegiate athletics may contribute to physiological stress, the observed changes in this study were more closely associated with travel-related circadian rhythm disruptions.

The significant increase in cortisol levels in the travel group compared to the non-travel group (Table 8, Table 9) aligns with evidence linking circadian misalignment to hypothalamic-pituitary-adrenal (HPA) axis activation,[4] and further supports a connection between eastward travel and bodily stress responses. Eastward travel in particular compresses daylight exposure, requiring phase advances in circadian timing, resulting in slower acclimation compared to the phase delays induced by westward travel.[19] This finding underscores the need for effective interventions to manage stress in traveling athletes, as heightened cortisol levels can impact metabolism, performance, and immune function.[24] Chronic stress elevates the risk of cardiovascular disease and related health issues, and recurrent acute stress exacerbates pre-existing conditions or impairs daily function.[24] The combination of sleep deprivation and the compression of daylight hours due to transmeridian air travel may significantly contribute to stress and cortisol dysregulation. These factors place notable strain on the suprachiasmatic nuclei (SCN) in the hypothalamus, which is responsible for regulating internal circadian rhythms.[6,25] As was observed in this study, disrupting the endogenous circadian rhythm can alter natural physiological patterns of cortisol secretion.[6] The frequent need for the body to adjust its internal clock may significantly contribute to heightened stress response and cortisol levels experienced by the traveling athletes, rather than the flights themselves. The large effect sizes in our cohort suggest that cortisol dysregulation may represent a key pathway through which travel affects athlete health and recovery.

Though no significant changes in total body fat percentage were observed in either group, significant increases in trunk body fat percentage and waist-to-hip ratio in the travel group indicate that regional fat distribution may be influenced by travel stress (Table 4, Table 5). The relationship between visceral adiposity and intermittent stressors observed in this study aligns with existing literature in which chronic circadian disruptions, particularly in shift work, are associated with increased visceral fat deposition. Experimental animal models of shift work revealed that chronic clock misalignment induced adipocyte hypertrophy, visceral fat accumulation, and inflammatory remodeling of adipose tissue linking circadian disruption, adiposity, and metabolic dysfunction.[26] Chronic and unpredictable stress can lead to preferential fat storage in mesenteric fat cells, promoting increased trunk visceral fat and waist circumference, as observed in the traveling athletes of this study.[27] It is important to observe this shift in fat distribution as visceral fat is an early indicator of metabolic syndrome and cardiovascular disease.[28] Evidence suggests a mechanism linking unpredictable stressors and visceral fat distribution.[28-29] Consequently, this physiological stress can affect the physical conditioning and performance of athletes, as well as increase their risk of developing long-term health issues.[30-31] Our findings are consistent with these reports and highlight that even in highly trained collegiate athletes, repeated disruption of circadian rhythms may predispose individuals to abnormal and unfavorable body composition changes with implications for long-term cardiometabolic risk. Investigating such stressors may be crucial to developing strategies to mitigate the effects of frequent travel.

A statistically significant time effect for leg bone mineral density (LBMD) was observed, driven by a modest increase in the travel group (Table 6). Such a pattern may indicate a potential adaptive response to the increased physical demands and stress associated with travel. However, this

positive adaptation lies outside the scope of the current study, and may reflect an interaction between travel-related stress and high-impact training demands contributing to this localized response. No significant changes, however, were observed in other regional bone mineral density measurements. This result suggests that though travel may not broadly impact bone health, localized effects warrant closer investigation.

A strength of this study was the inclusion of a non-traveling control group from the same athletic program, who engaged in supervised training sessions and local practice games, naturally providing an intensity control. This reduced the likelihood that differences were derived from workload disparities rather than travel exposure. However, objective workload measures (e.g., GPS tracking, session-RPE) were not collected, limiting precise comparisons of training load between groups. Additional limitations include the available sample size, limited sample diversity, and lack of control over non-sports related travel. Although the sample size was modest, moderate-to-large effects were observed in cardiovascular parameters and cortisol, suggesting that the study had adequate sensitivity to detect meaningful group differences in these primary outcomes. Smaller effect sizes in anthropometric and bone density measures, however, may require larger samples to clarify whether subtle changes occur in response to travel. Additionally, the sample was composed of healthy, college-aged female athletes, which may not accurately represent the larger population, contributing to a lack of sample diversity. Air-based leisure travel was not formally recorded, and therefore we cannot fully exclude its potential influence. Nonetheless, given the demanding and consistent training and competition schedules, such travel was likely minimal and unlikely to meaningfully confound the observed results. Finally, measures were not taken to observe cortisol levels and secretion patterns in participants after the postseason sample collection, which limits insight into longer-term recovery.

The results of this study highlight the physiological impacts of frequent transmeridian time-zone crossings on the health of female collegiate volleyball players. These findings emphasize the need to consider intervention development to manage travel-related stress and mitigate its potential effects on athletes' cardiovascular health. Strategies such as circadian phase-shifting protocols, structured sleep interventions, or tailored recovery programs, as described in previous studies,[32] may help mitigate cardiovascular strain and cortisol dysregulation. Future studies should incorporate objective workload monitoring and extend observations into a postseason period to capture recovery trajectories. Larger, more diverse samples may be essential to confirm whether observed patterns generalize to broader athletic and non-athletic populations, thereby enhancing applicability and clarifying the long-term health implications of repeated circadian disruption.

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